Analysis and analytical characterization of bioheat transfer during radiofrequency ablation

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\textbf{Abstract}
Understanding thermal transport and temperature distribution within biological organs is important for therapeutic aspects related to hyperthermia treatments such as radiofrequency ablation (RFA). Unlike surface heating, the RFA treatment volumetrically heats up the biological media using a heating probe which provides the input energy. In this situation, the shape of the affected region is annular, which is described by an axisymmetric geometry. To better understand the temperature responses of the living tissues subject to RFA, comprehensive characteristics of bioheat transport through the annular biological medium is presented under local thermal non-equilibrium (LTNE) condition. Following the operational features of the RFA treatment, based on the porous media theory, analytical solutions have been derived for the blood and tissue temperature distributions as well as an overall heat exchange correlation in cylindrical coordinates. Our analytical results have been validated against three limiting cases which exist in the literature. The effects of various physiological parameters, such as metabolic heat generation, volume fraction of the vascular space, ratio of the effective blood to tissue conductivities, different biological media and the rate of heat exchange between the lumen and the tissue are investigated. Solutions developed in this study are valuable for thermal therapy planning of RFA. A criterion is also established to link deep heating protocol to surface heating.

\section{Introduction}
The modeling of bioheat transfer has been employed extensively in medical thermal therapeutic applications for predicting the temperature distribution (Khaled and Vafai, 2003; Khanafer and Vafai, 2009). Nowadays, cancer is still one of the lowest survival rate diseases. Hyperthermia treatment such as radiofrequency ablation (RFA) (Peng et al., 2011), microwave, laser (Dombrovsky et al., 2012), magnetic fluid (Giordano et al., 2010), etc. is recognized as the fourth adjunct cancer therapy technique following surgery, chemotherapy and radiation techniques. When biological tissues are subjected to high temperatures, which are typically 40–45°C (Field, 1987), heat shock can cause a cancer cell to lose viability and eventually induce cell death (Kiss et al., 2009). In contrast to resection techniques, which have a poor prognostic outcome, RFA has the potential to improve and to optimize clinical treatment with fewer side effects (Goldberg et al., 2000; Peng et al., 2011). Similar to cryotherapy (Chua et al., 2007), RFA also has a minimally invasive nature. Briefly, RFA induces resistive heating in tissues in direct contact with an ablation electrode (Boronyak and Merryman, 2014). Liu (2001) presented an analytical solution to the Pennes bioheat transfer equation in three-dimensional geometry with practical hyperthermia boundary conditions and random heating. Chung and Vafai (2014) investigated analytically and numerically the effects of hyperthermia on low-density lipoprotein transport and heat transfer within a multi-layered arterial wall accounting for the fluid–structure interaction. However, the thermal responses of a living organ under ablation treatments have not yet been fully evaluated quantitatively in the clinical field. So it is imperative to study the general characteristics of bioheat transfer with medical affiliates in order to demonstrate the relationship between the heating power deposited in the tissue and the resulting tissue status post treatment.

A biological tissue consists of a microvascular bed with blood flow through many vessels. As such it is quite natural to treat the living tissue as a porous medium (Khaled and Vafai, 2003; Khanafer and Vafai, 2006; Zhang, 2009). Thus, the porous media theory can be utilized for bioheat transfer analysis, in which fewer assumptions are needed as compared to other established bioheat transfer models (Khaled and Vafai, 2003; Khanafer and Vafai, 2006, 2009; Nakayama and Kuwahara, 2008; Mahjoob and Vafai, 2009, 2010). Two primary models for analyzing heat transfer in a porous medium are: local thermal equilibrium (LTE) and local thermal non-equilibrium (LTNE). The LTE model is based on the assumption that the temperature for tissue phase is equal to that for blood phase, on a local basis, everywhere inside the porous medium. However, the LTE model does not hold for some physical characteristics of bioheat transfer with medical affiliates in order to demonstrate the relationship between the heating power deposited in the tissue and the resulting tissue status post treatment.
situations when the temperature difference between the two phases is not negligible (Khaled and Vafai, 2003). In such cases, the LTNE model should be utilized to investigate the blood temperature changes as a result of tissue-blood convective heat exchange and blood perfusion (Xuan and Roetzel, 1997; Lee and Vafai, 1999; Alazmi and Vafai, 2000; Zhang, 2009; Mahjoob and Vafai, 2009, 2010; Rattanadecho and Keangin, 2013). Mahjoob and Vafai (2009) carried out a comprehensive investigation of bioheat transport through the tissue/organ incorporating hyperthermia treatment using LTNE, and had established exact solutions for the blood thermal conductivity ratio, metabolic heat generation, etc. is analyzed. A criterion is also constructed to bridge the bioheat transfer for deep heating such as RFA and that for surface heating (Mahjoob and Vafai, 2009, 2010) through a geometrical analysis.

2. Mathematical modeling

2.1. Problem description

Biological tissue generally contains blood vessels, cells and interstitial space (Mahjoob and Vafai, 2009), which can be categorized as vascular and extra-vascular regions, as shown in Fig. 1(a). As such, the whole anatomical structure can be modeled as a porous matrix through which the blood infiltrates. Generally, the pressure is uniformly higher throughout the tumor as compared to the peripheral values, which leads to an extremely slow interstitial flow. Hence, the blood flow within the tumor region can be represented by the Darcy flow model (Wu et al., 2009; Mahjoob and Vafai, 2009, 2010; Coolsen et al., 2012). In this study, hydraulically and thermally fully developed condition is assumed. The flow is steady and incompressible. Natural convection and radiation

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are negligible and thermodynamic properties of the blood and tissue are considered to be temperature-independent.

Farris et al. (2011) proposed the mathematical model for predicting the human achilles tendon body core temperature during running. The RFA treatment region can be represented by an annulus with an imposed heat flux at the inner surface (the interface between the probe and the organ) and a body core temperature at the outer surface (the edge of the heated region). Fig. 1(b) illustrates a typical axisymmetric biological model including an ablation electrode (e.g. a heating probe) with a heat source along its shank. As seen, the cylindrical probe with a radius \( R_i \), which is bounded by an annular treatment region, produces a constant heat flux \( Q_{\text{met}} \) along the shank.

### 2.2. Governing equations

The anatomic structure is modeled as a porous medium consisting of the blood and tissue (solid matrix) phases. The governing energy equations for both phases incorporating internal heat generation (e.g. metabolic reactions) and LTNE condition can be represented as

**Blood phase:**

\[
k_{b,\text{eff}} \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial T_b}{\partial r} \right) + h_i \rho_b c_b \left( T_b - T_i \right) = \frac{\varepsilon_c \rho_b c_b}{\rho_b c_b} \frac{\partial H}{\partial t} \]

**Tissue phase:**

\[
k_{t,\text{eff}} \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial T_t}{\partial r} \right) - h_i \rho_b c_b \left( T_b - T_t \right) + \left(1 - \varepsilon \right) Q_{\text{met}} = 0
\]

where \( k_{b,\text{eff}} = \varepsilon_b k_b + k_{b,\text{dis}} \)

\[
k_{t,\text{eff}} = (1 - \varepsilon) k_{t,\text{dis}}
\]

where \( r \) is the radial coordinate, \( T_b \), \( T_t \), \( k_{b,\text{eff}} \), \( k_{t,\text{eff}} \), \( k_{b,\text{dis}} \), \( \varepsilon_b \) and \( \varepsilon \) are the intrinsic phase average blood and tissue temperatures, blood and tissue effective thermal conductivities, blood dispersion thermal conductivity, porosity (the volume fraction of the vascular space), blood density and specific heat, respectively. The blood–tissue interfacial heat transfer coefficient is represented by \( h_i \), and the specific surface area by \( a_{sb} \) and \( Q_{\text{met}} \) is the metabolic heat generation.

### 2.3. Boundary conditions

Based on established results under LTNE condition in the literature (Lee and Vafai, 1999; Maralae and Vafai, 2001; Mahjoob and Vafai, 2009; Yang and Vafai, 2010), the imposed heat flux at the surface of heating probe can be represented as

\[
q_w = -k_{b,\text{eff}} \frac{\partial T_b}{\partial r} \bigg|_{r = R_i} - k_{t,\text{eff}} \frac{\partial T_t}{\partial r} \bigg|_{r = R_i}
\]

The temperature at the interface between the heating probe and the body organ is likely to be uniform regardless of whether it contacts the tissue solid matrix or the blood. As such, the tissue and blood temperatures at the probe–organ interface will be the same (Lee and Vafai, 1999; Maralae and Vafai, 2001; Mahjoob and Vafai, 2009, 2010).

\[
(T_b)_{r = R_i} = (T_t)_{r = R_i} \approx T_w
\]

The external heat flux generated by the heating probe influences the organ within a penetration depth of \( H \). Here, \( H = R_o - R_i \). On the outer surface the tissue and blood temperatures will be the same at the body core temperature at 37 °C (Giordano et al., 2010; Peng et al., 2011).

\[
(T_b)_{r = R_o} = (T_t)_{r = R_o} \approx T_c = 37^\circ C
\]

### 2.4. Normalization

To normalize the governing equations and boundary conditions, the dimensionless variables are introduced as follows

\[
n = \frac{r}{L}, \quad \eta_1 = \frac{\varepsilon_c r}{c_c H}, \quad \eta_2 = \frac{1 - \varepsilon}{\varepsilon_b}, \quad \eta = \frac{L}{R_o}, \quad \kappa = \frac{k_{b,\text{eff}}}{k_{t,\text{eff}}}, \quad \text{Bi} = \frac{k_{b,\text{eff}} H}{\varepsilon_c c_c H}
\]

where \( \kappa \) is the ratio of the effective thermal conductivity of the blood to tissue and Bi the Biot number.

Adding the governing Eqs. (1) and (2), integrating the resultant equation over the cross-sectional area and applying boundary conditions given by Eqs. (5) and (7) leads to the following relationship

\[
\frac{\varepsilon_c \rho_b c_b}{\rho_b c_b} \left( \frac{d(T_b)}{dt} \right) = \frac{2(n + \Gamma) Q_{\text{met}}}{(1 + n) H} \left(1 - \varepsilon \right)
\]

where the derivative \( \frac{d(T_b)}{dt} \) is constant for thermally fully-developed flow, and

\[
\Gamma = \frac{1}{Q_{\text{met}}} \left( k_{b,\text{eff}} \frac{d(T_b)}{dr} + k_{t,\text{eff}} \frac{d(T_t)}{dr} \right) \bigg|_{r = R_i}
\]

Considering the Darcy flow model and substituting Eqs. (8) and (9), the governing Eqs. (1) and (2) as well as boundary conditions (5) and (7) can be rewritten as

\[
\frac{1}{n \eta} \frac{\partial}{\partial \eta} \left( \frac{\partial T_b}{\partial \eta} \right) + \Phi (\theta_b - \theta_o) = \frac{2(n + \Gamma)}{1 + n} \theta_c
\]

\[
\frac{\partial}{\partial \eta} \left( \frac{\partial T_t}{\partial \eta} \right) - \Phi (\theta_t - \theta_o) + \Phi = 0
\]

\[
\theta_b \big|_{a = a_o} = \theta_t \big|_{a = a_o} = 0
\]
\[ \theta_b \big|_{t = \nu} = \theta_i \big|_{t = \nu} = \theta_c \]

where \( \theta_c \) is the dimensionless core body temperature such that

\[ \theta_i = \frac{k_{i,eff}(T_c - T_w)}{q_w H} \quad \text{(15)} \]

3. Analytical solution

3.1. Blood and tissue temperature distributions

The governing Eqs. (11) and (12) are solved analytically subject to the constant heat flux \( q_w \) at the inner surface and the constant temperature \( T_c \) at the outer surface. As such, the obtained blood and tissue temperature distributions may be employed to rewrite Eq. (10). In other words, the analytical solution to Eqs. (11) and (12) is independent of the term \( \Gamma \) (Mahjoo and Vafai, 2011). Following Mahjoo and Vafai (2011), the two governing Eqs. (11) and (12) are added to yield a new equation

\[ \frac{1}{\eta} \frac{\partial}{\partial \eta} \left[ \eta \frac{\partial}{\partial \eta} (\kappa \theta_b + \theta_t) \right] = \frac{2(n + \Gamma)}{1 + n} \quad \text{(16)} \]

which is the second order Euler–Cauchy equation with the dependent variable \( (\kappa \theta_b + \theta_t) \), which can be solved with the modified boundary conditions based on Eqs. (13) and (14) as

\[ (\kappa \theta_b + \theta_t) \big|_{\eta = \eta_0} = 0 \quad \text{(17)} \]

\[ (\kappa \theta_b + \theta_t) \big|_{\eta = \eta_i} = (1 + \varepsilon) \theta_c \quad \text{(18)} \]

The complete solution for Eq. (16) is found to be

\[ \kappa \theta_b + \theta_t = C_1 + C_2 \ln \eta + \frac{(n + \Gamma) \eta^2}{2(1 + n)} - \kappa \theta_b \quad \text{(19)} \]

where \( C_1 \) and \( C_2 \) denote the unknown constants illustrated in Appendix A [Eqs. A.1–A.6].

It is worth noting that \( \theta_t \) on the outer surface is still unknown. The normalized form of Eq. (10), based on Eq. (8), can be written as

\[ \Gamma = \frac{\partial \theta_b}{\partial \eta} + \frac{\partial \theta_t}{\partial \eta} \big|_{\eta = \eta_0} \quad \text{(19)} \]

Then, the solution for \( \Gamma \) can be easily obtained by solving Eq. (21), as illustrated in Appendix A [Eqs. A.7–A.9]. Rewriting Eq. (19) as

\[ \theta_t = C_1 + C_2 \ln \eta + \frac{(n + \Gamma) \eta^2}{2(1 + n)} - \kappa \theta_b \quad \text{(22)} \]

and substituting it into Eq. (11) produces

\[ \frac{1}{\eta} \frac{\partial}{\partial \eta} \left( \eta \frac{\partial \theta_b}{\partial \eta} \right) - \lambda^2 \theta_b = -\frac{B_l}{k} \left[ C_1 + C_2 \ln \eta + \frac{(n + \Gamma) \eta^2}{2(1 + n)} \right] + \frac{1}{\kappa} \left[ rac{2(n + \Gamma)}{1 + n} - \Phi \right] \quad \text{(23)} \]

Eq. (23) is a modified Bessel equation. As such, we have decoupled Eqs. (11) and (12). Eq. (23) becomes a non-homogeneous ordinary differential equation which can be solved subject to the boundary conditions (13) and (14). As such, the complete solution takes the following form

\[ \theta_b = D_1 I_0(\lambda \eta) + D_2 K_0(\lambda \eta) + D_3 \ln \eta + D_4 \eta^2 + D_5 \quad \text{(24)} \]

where \( \lambda = \sqrt{(1 + \kappa)B_l / \kappa} \) and \( I_0 \) and \( K_0 \) are the zeroth order modified Bessel functions of the first and second kind respectively. Substituting Eq. (24) into the boundary conditions given by Eqs. (13) and (14), one can obtain the unknown constants \( D_1, D_2, D_3, D_4 \), and \( D_5 \) illustrated in Appendix A [Eqs. (A.10)–(A.24)]. It can be seen from Eqs. (A.1), (A.2) and (A.10)–(A.14), that all the constants are functions of the dimensionless core body temperature \( \theta_c \).

3.2. Determination of the dimensionless core body temperature

Once the dimensionless core body temperature \( \theta_c \) on the outer surface is known, the expressions of \( \Gamma, C_1, C_2, D_1, D_2, D_3, D_4 \) and \( D_5 \) are explicitly obtained. For flow through an annular biological medium, the dimensionless bulk mean blood temperature averaged over the cross section is calculated as

\[ \theta_{b,m} = \frac{2}{\eta_0^2 - \eta_i^2} \int_{\eta_i}^{\eta_0} \theta_b d\eta = \alpha_1 + \alpha_2 \theta_c \quad \text{(25)} \]

where \( \alpha_1 \) and \( \alpha_2 \) are given in Appendix A [Eqs. A.25–A.30].

Integrating both sides of Eq. (9) simultaneously with regard to \( z \) results in

\[ T_b = \frac{2n + r_q}{2} \left( 1 - \varepsilon \right) \frac{Q_{net}}{\rho b c_b \theta_c} + \frac{q_w H}{k_{eff}} (\theta_j - \theta_m) + T_e \quad \text{(26)} \]

The dimensionless bulk mean blood temperature can be defined as

\[ T_{b,m} = \frac{2}{K_0^2 - K_1^2} \int_{K_1}^{K_0} T_b dK = \beta_1 + \beta_2 \theta_c \quad \text{(27)} \]

where \( \beta_1 \) and \( \beta_2 \) are given in Appendix A [Eqs. A.31 and A.32].

From Eq. (15), we can obtain an expression for the dimensionless temperature at the probe–organ interface as

\[ T_w = T_c - \frac{q_w H}{k_{eff}} \theta_c \quad \text{(28)} \]

Similarly,

\[ T_w = T_{b,m} - \frac{q_w H}{k_{eff}} \theta_{b,m} \quad \text{(29)} \]

Substitution of Eqs. (25), (27) and (28) into Eq. (29) leads to

\[ T_c - \frac{q_w H}{k_{eff}} \theta_c = \beta_1 + \beta_2 \theta_c - \frac{q_w H}{k_{eff}} (\alpha_1 + \alpha_2 \theta_c) \quad \text{(30)} \]

Consequently, the dimensionless core body temperature \( \theta_c \) can be obtained by solving Eq. (30) resulting in

\[ \theta_c = \frac{q_w H \theta_1 + k_{eff}(T_c - \theta_1)}{q_w H (1 - \alpha_2) + k_{eff} \beta_2} \quad \text{(31)} \]

3.3. Heat transfer correlations

The probe–organ interface transfer coefficient for the local thermal non-equilibrium model is obtained from

\[ h_w = \frac{q_w}{T_w - T_{b,m}} \quad \text{(32)} \]

After solving for the blood and tissue temperatures, the local Nusselt number, \( Nu \), can be obtained from

\[ Nu = \frac{h_w (2H)}{k_{eff}} = -\frac{2}{\kappa \theta_{b,m}} \quad \text{(33)} \]

4. Results and discussion

4.1. Validation

Our analytical solutions are validated against very few existing exact solutions for the following three limiting cases: a parallel-plate channel subject to a heat flux on one surface and uniform core temperature on the other (Limiting case I) surface (Mahjoo and Vafai, 2009, 2010) or when subject to a heat flux on both
(Limiting case II) surfaces (Mahjoob and Vafai, 2009, 2010), and an annulus subject to a heat flux on the inner surface and an adiabatic condition on the outer (Limiting case III) surface (Qu et al., 2012). It should be noted that all the results are obtained at the entrance, i.e., z = 0 unless otherwise stated.

4.1.1. Limiting case I ($\psi \rightarrow \infty, \Gamma \neq 0$)

Let us define $\psi = R_i / H$ as the aspect ratio of the annular geometry under consideration. As $\psi \rightarrow \infty$, the analytical solution within the annulus approaches that within the planar geometry (Mahjoob and Vafai, 2009, 2010). For purpose of plotting the results, typical values for the thermophysical properties are taken as: $k_b = 0.45 \text{ W/m}^\circ\text{C}$, $k_t = 0.54 \text{ W/m}^\circ\text{C}$, $c_b = 3960 \text{ J/kg}^\circ\text{C}$ and $\rho_b = 1058 \text{ kg/m}^3$. The blood velocity and temperature at the entrance, metabolic heat generation, the imposed heat flux and the blood dispersion thermal conductivity are set to be $u_e = 0.02 \text{ m/s}$, $T_e = 37 \circ\text{C}$, $Q_{\text{met}} = 4200 \text{ W/m}^3$, $q_w = 200 \text{ W/m}^2$ and $k_{\text{disp}} = 0.006 \text{ W/m}^\circ\text{C}$, respectively. This yields $\Phi = 0.189$ and $\kappa = 0.105$. A representative volume fraction ($\varepsilon = 0.1$) of the vascular system is employed for some of the comparisons (Mahjoob and Vafai, 2009). For the validation with the planar case, $R_i = 10 \text{ m}$ and $R_o = 10.01 \text{ m}$ are utilized, thus $H = 0.01 \text{ m}$ and $\psi = 1000$, to setup the present model to have approximately the same geometry and boundary conditions as that utilized by Mahjoob and Vafai (2009, 2010). As shown in Fig. 2(a), the present blood and tissue temperature distributions coincide very well with the results derived by Mahjoob and Vafai (2009, 2010).

4.1.2. Limiting case II ($\psi \rightarrow \infty, \Gamma = 0$)

As mentioned in Mahjoob and Vafai (2011), $\Gamma$ is represented by Eq. (10) for a constant temperature boundary condition while $\Gamma$ is zero for an adiabatic boundary condition. To compare the present solution with the results for the limiting case II, herein we set $\Gamma = 0$ and replace Eq. (14) by the following adiabatic boundary condition

$$\frac{\partial \theta}{\partial \eta} \bigg|_{\eta = \eta_o} = 0$$

Using a similar approach as was used in deriving Eqs. (22) and (24), the solutions for Eqs. (11) and (12) subject to boundary conditions (13) and (34) are readily obtained, where are in the same form as those given by Eqs. (22) and (24), respectively. However, the involved constants $C_1$, $C_2$, $D_1$, $D_2$, $D_3$ and $D_5$ should be replaced with $C_01$, $C_02$, $D_01$, $D_02$, $D_03$ and $D_05$, which are given in Appendix A [Eqs. A.33–A.39]. For comparison purpose, the same geometric parameters as previous limiting case were used. Fig. 3(b) and (c) display the comparison for blood and tissue temperature distributions obtained by current analytical results with the ones obtained by Mahjoob and Vafai (2009, 2010). Once again, an excellent agreement can be observed. Also, the validation

\begin{align*}
\text{Fig. 2. Comparison of the present analytical solutions for (a) temperature (Limiting case I, $\kappa = 0.105$), (b) temperature (Limiting case II, $\kappa = 0.1$), (c) temperature (Limiting case II, $\kappa = 10$), and (d) Nusselt number (Limiting cases I and II) with the results of Mahjoob and Vafai (2009, 2010).}
\end{align*}
of Nusselt number for both limiting cases I and II is depicted in Fig. 2(d).

4.1.3. Limiting case III (\(Da = 0, \Gamma = 0\))

As the last limiting case, we consider an annulus with inner and outer radii \(R_i = 0.01 \text{ m} \) and \(R_o = 0.03 \text{ m} \). As the Darcy number \(Da \to 0\), the Brinkman–Darcy extended flow model tends to Darcy flow model (Hooman and Ranjbar-Kani, 2004). To compare the present analytical solution with the work of Qu et al. (2012), the Darcy number is assumed to be small enough, say \(Da = 10^{-10}\). Fig. 3(a) and (b) shows the comparison of present temperature distributions with the ones given by Qu et al. (2012). Fig. 3(c) depicts the validation of the Nusselt number. All figures display an excellent agreement between the current results and those by Qu et al. (2012).

4.2. Effect of physical parameters on the temperature distributions

In what follows, based on physiological data, the inner and outer radii of the treatment region are taken as \(R_i = 3 \times 10^{-5} \text{ m} \) and \(R_o = 5 \times 10^{-3} \text{ m} \). Fig. 4(a) illustrates the effect of metabolic heat generation on the blood and tissue temperature distributions for \(\Phi = 0.01, 0.1 \) and 0.5. The energy required for replenishment is produced by the oxidation of the nutrients supplied to the biological tissues. Any decrease in metabolic heat has to be compensated by an increase in the input energy to attain hyperthermia and vice versa. As expected, larger heat generation rate results in higher temperature in the tissue as well as the blood within it. Also, an increase in the metabolic heat generation rate enhances the temperature difference between the blood and tissue phases.

The vascular porosity, which increases with age (Norman et al., 2008), exhibits a wide range of values in human tissue, from 4% to more than 16% (Cardoso et al., 2013). Fig. 4(b) shows the effect of vascular volume fraction on the blood and tissue temperature distributions for \(\varepsilon = 0.05, 0.1 \) and 0.3. An increase in the volume fraction results in a more uniform temperature distribution, which possibly leads to a more effective hyperthermia treatment. It is worth noting that a change in the vascular volume fraction also translates in a change in the blood and tissue effective thermal conductivities. The natural body thermal regulation system increases or decreases the vascular volume fraction of the biological organ when exposed to a higher or lower temperature. This phenomenon has also been reported for surface heating by Mahjoob and Vafai (2009). It should be pointed out that the blood and tissue temperatures are within a relatively small range in certain physiological or medical situations. Therefore, the thermal properties of the biological bodies can be considered as constants.

Fig. 4(c) demonstrates the effect of the Biot number on the blood and tissue temperature distributions for \(Bi = 0.01, 0.5, 10 \) and 50. It can be observed from this figure that the blood and tissue temperatures and their difference increases with a decrease in the Biot number. The reason is that when Bi is small, the internal heat

![Fig. 3. Comparison of the present analytical solutions for (a) temperature (Limiting case III, \(\kappa = 0.1\)), (b) temperature (Limiting case III, \(\kappa = 10\)), and (c) Nusselt number (Limit case III) with the results of Qu et al. (2012).](image-url)
transfer between the blood and tissue phases becomes relatively weak. Therefore, the blood perfusion cannot effectively remove the supplied energy during the probe based thermal therapy. In other words, smaller Bi is clinically helpful for improving the hyperthermia treatment effect.

Fig. 5(a) shows the impact of the effective thermal conductivity ratio on the blood and tissue temperature distributions for \( \kappa = 0.01, 0.1, 1 \) and 10. A decrease in the effective thermal conductivity ratio results in an increase in the blood and tissue temperatures, due to a subsequent decrease in heat exchange between the probe surface and the blood. The analytical results presented in this study are applicable to thermal problems within several biological organs. Typical thermophysical properties of biological media (Bauman et al., 2004; He et al., 2008; Gilbert et al., 2009; Wessapan et al., 2011, 2012; Wessapan and Rattanadecho, 2012) are listed in Table 1. Here, the blood thermal dispersion effect can be represented by \( k_b = 0.1 \Pr Re_b \) (Amiri and Vafai, 1994). This yields \( \kappa = 1.5008, 0.2765, 1.2860, 3.3564 \) and 0.5742, respectively, for Liver, Brain, Cornea, Bone and Fat considered in our study. Fig. 5(b) displays the results incorporating the blood thermal dispersion for \( Bi = 10 \) and \( \Phi = 0.1 \). It is apparent that the blood and tissue temperatures within the Brain are higher than those within the other four biological media. This implies that the therapeutic heating can be more readily achieved within the Brain. As a point of comparison, the results without accounting for the blood thermal dispersion are also shown in the figure. When the thermal dispersion is not accounted for, the effective thermal conductivity ratio changes to \( \kappa = 1.3582, 0.2103, 1.1638, 0.4190 \) and 3.0963 for the five biological media. As seen in Fig. 5(b), thermal dispersion reduces the blood and tissue temperatures, which translates into more required input energy to compensate for the decreased temperature arising from the blood dispersion.

Fig. 5(c) depicts the variation of blood and tissue temperature distributions at four axial locations along the \( z \)-axis with \( \xi = z / H = 0, 10, 50 \) and 100. As expected, the temperature difference between blood and tissue reduces as \( \xi \) increases. When \( \xi > 0 \), especially when \( \xi \geq 50 \), the temperature for both phases for \( \zeta \leq 0.6 \) declines while that for \( \zeta > 0 \) illustrates an opposite trend. When the probe shank is long enough, the temperatures in the vicinity of the probe surface will be relatively low while those near a healthy tissue will be relatively high. In such a situation, the tumor cells may not be efficiently treated but the normal tissue may be damaged due to the high temperature.

4.3. Establishing the criterion for simulation of a planar geometry to represent an annular one

As discussed earlier, if the aspect ratio \( \psi \) is set to be large enough the results for an annulus will match those obtained by the planar geometry. Here we seek to obtain the criterion for when the results obtained for the two geometries will match. For this purpose, the thickness of the annulus is kept unchanged, say \( H = 0.01 \) m while the inner radius \( R_i \) varies starting from one times the thickness. In order to assess the matching of annular to planar
geometries, the following criterion is employed

\[ E = \left| \frac{\theta_a - \theta_p}{\theta_p} \right| \times 100\% \rightarrow 1\% \]  

(35)

where \( \theta_a \) and \( \theta_p \) are the dimensionless temperatures in the annular and planar geometries respectively and \( E \) is the relative error obtained by analytical temperatures in the annular and planar geometries.

The calculated results for \( E \) in the present study and Eqs. (33) and (34) of Mahjoob and Vafai (2009) are tabulated for two locations along the \( r \)-axis. In Table 2, the first data corresponding to each \( \psi \) is for the blood phase and the second one is for the tissue phase. It is evident from this table that the relative error
decreases with an increase in $\psi$. The maximum relative errors corresponding to $\psi=150$ and $200$ are 1.038% and 0.779% respectively. Moreover, it is found that the value of the relative error decreases linearly from $\psi=100$, 150 to 200. Therefore, the minimum aspect ratio $\psi_{\text{min}}=157$ can be obtained by linear interpolation between $\psi=150$ and 200. Thus, a relationship between annular and planar geometries has been constructed through the aspect ratio of the annulus. This implies the similarity between deep heating and surface heating in certain conditions.

5. Conclusions

Understanding and predicting heat transport and temperature distribution inside biological media are crucial in hyperthermia treatments such as RFA. In this work, analytical solutions are established for the blood and tissue temperature distributions as well as the overall heat exchange correlations. Our analytical results were comprehensively validated. The derived analytical solutions provide general characteristics of the bioheat transfer during a RFA treatment. The effects of physiological parameters such as metabolic heat generation, vascular volume fraction, etc., were analyzed in this work. An increase in the metabolic heat generation or in the vascular volume fraction enhances the temperatures for the blood and tissue phases. When $\zeta > 0$, especially when $\zeta \geq 5$, the blood and tissue temperatures for $\zeta < 0.6$ decline while those for $\zeta > 0.6$ illustrate an opposite trend. The obtained analytical results enable the clinicians to predict the temperature distribution in a biological medium within a real time feedback mechanism. Based on the present solutions, an approach to optimize the hyperthermia parameters can be obtained for the tumor destroying temperature and tissue burn threshold. Finally, a criterion for simulation of a planar geometry to represent an annular one is established.

As reported by many investigators, the mechanical properties of treated tissues will change after a thermal therapy and, in turn, affect the post-treatment structural integrity and functions of the tissue (Chen and Humphrey, 1998; Qian et al, 2013, Lin et al. (2012) found that the Young’s modulus and stiffness of skin tissue decreased with increasing heating temperature, which was correlated to the skin microstructure changes induced by thermal denaturation. Therefore, the present analytical results enable the researchers to better understand the effect of post-treatment of treated tissues or organs on their structural integrity and hence more effectively design the clinical therapy protocols.

Conflict of interest statement

None.

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Appendix A

The constants involved in Eq. (19) are given by

$$C_1 = C_{11} + C_{12} \theta_c$$  \hspace{1cm} (A.1)

$$C_2 = C_{21} + C_{22} \theta_c$$  \hspace{1cm} (A.2)

where

$$C_{11} = \frac{n + F_1}{2(1 + n)} \frac{n \eta_0 - \eta_i}{\ln \eta_i - \ln \eta_0}$$  \hspace{1cm} (A.3)

$$C_{12} = \frac{1}{\ln \eta_i - \ln \eta_0} \left[ (1 + k) \ln \theta_c + \frac{F_2}{2(1 + n)} \left( \eta_i^2 \ln \eta_i - \eta_0^2 \ln \eta_0 \right) \right]$$  \hspace{1cm} (A.4)

$$C_{21} = \frac{n + F_1}{2(1 + n)} \left( \eta_i^2 - \eta_i^2 \right)$$  \hspace{1cm} (A.5)

$$C_{22} = \frac{1}{\ln \eta_i - \ln \eta_0} \left[ -(1 + k) + \frac{F_2}{2(1 + n)} \left( \eta_i^2 - \eta_i^2 \right) \right]$$  \hspace{1cm} (A.6)

The expression for $\Gamma$ is given by

$$\Gamma = \Gamma_1 + \Gamma_2 \theta_c$$  \hspace{1cm} (A.7)

where

$$\Gamma_1 = \frac{n(1 + 2 \ln \eta_i - 2 \ln \eta_0) \eta_i^2 - \eta_i^2}{\eta_0^2 + \eta_i^2}$$  \hspace{1cm} (A.8)

$$\Gamma_2 = \frac{2(1 + k)(1 + n)}{2(1 + k)(1 + n)}$$  \hspace{1cm} (A.9)

The constants $D_1$, $D_2$, $D_3$, $D_4$ and $D_5$ involved in Eq. (24) are given by

$$D_1 = D_{11} + D_{12} \theta_c$$  \hspace{1cm} (A.10)

$$D_2 = D_{21} + D_{22} \theta_c$$  \hspace{1cm} (A.11)

$$D_3 = D_{31} + D_{32} \theta_c$$  \hspace{1cm} (A.12)

$$D_4 = D_{41} + D_{42} \theta_c$$  \hspace{1cm} (A.13)

$$D_5 = D_{51} + D_{52} \theta_c$$  \hspace{1cm} (A.14)

where

$$D_{11} = \frac{K_0(\lambda \eta_0)(D_{11} \ln \eta_i + D_{41} \eta_i^2 + D_{51}) - K_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51})}{I_0(\lambda \eta_0)K_0(\lambda \eta_i) - I_0(\lambda \eta_i)K_0(\lambda \eta_0)}$$  \hspace{1cm} (A.15)

$$D_{12} = \frac{K_0(\lambda \eta_0)(D_{11} \ln \eta_i + D_{41} \eta_i^2 + D_{51}) - K_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51} - 1)}{I_0(\lambda \eta_0)K_0(\lambda \eta_i) - I_0(\lambda \eta_i)K_0(\lambda \eta_0)}$$  \hspace{1cm} (A.16)

$$D_{21} = \frac{-I_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51} + I_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51})}{I_0(\lambda \eta_0)K_0(\lambda \eta_i) - I_0(\lambda \eta_i)K_0(\lambda \eta_0)}$$  \hspace{1cm} (A.17)

$$D_{22} = \frac{-I_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51} + I_0(\lambda \eta_0)(D_{31} \ln \eta_i + D_{41} \eta_i^2 + D_{51} - 1)}{I_0(\lambda \eta_0)K_0(\lambda \eta_i) - I_0(\lambda \eta_i)K_0(\lambda \eta_0)}$$  \hspace{1cm} (A.18)

$$D_{31} = \frac{C_{21}}{1 + k}$$  \hspace{1cm} (A.19)

$$D_{32} = \frac{C_{22}}{1 + k}$$  \hspace{1cm} (A.20)

$$D_{41} = \frac{n + F_1}{2(1 + k)(1 + n)}$$  \hspace{1cm} (A.21)

$$D_{42} = \frac{F_2}{2(1 + k)(1 + n)}$$  \hspace{1cm} (A.22)

$$D_{51} = \frac{C_{11}}{1 + k} + \frac{4D_{41}}{\lambda^2} - \frac{1}{(1 + k)B} \left[ \frac{2(n + F_1)}{1 + n} + \phi \right]$$  \hspace{1cm} (A.23)
The new values of the constants given in Eqs. (A.1), (A.2) and (A.10)–(A.14).

References


